ARTERIAL HYPERTONUS AND ITS RELATION TO HIGH SPHYGMOMANOMETER READINGS.

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By

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Before discussing the condition of Hypertonic Contraction of the arteries and its relation to high Sphygmomanometer readings, it would be well to give a short resumé of the modern theories of arterial thickenings.

Considerable diversity of opinion exists, not only as regards the aetiology of vascular thickenings, a subject it is not proposed to discuss here, but also as regards their pathology.

The terms "Atheroma" and Arteriosclerosis" seem to be used indiscriminately in many works. Sir Clifford Allbutt in his "System of Medicine" gives Dr. Mott's views on the subject. Under the section entitled "Arteriosclerosis" he describes both nodular and diffuse forms. He gives as a definition "a local or general thickening of the arterial wall with loss of contractility and elasticity, occasioned by fibrous overgrowth mainly of the tunica intima, secondary and proportional to degeneration of the muscular and elastic elements of the media." As will be shown later, "loss of contractility and elasticity" does not always occur, and where contractility and elasticity are retained, the muscular elements of the media must be present to a greater or less extent.

Professor Wm. Russell,[1] on the other hand, speaks of/

of the nodular form as "atheroma" and limits the term "arteriosclerosis" to the diffuse variety.

All seem agreed as to the pathology of "Atheroma," or, as some authors call it, "endarteritis deformans." It commences as a localised overgrowth of the layer of connective tissue situated between the endothelial lining of the vessel and the internal elastic lamina. This overgrowth quickly passes on to a process of degeneration, the degenerated areas in many cases becoming the seat of calcareous deposits. Some authors, e.g., Paul, Monheberg, and Bunting, describes the formation of true bone in these patches. The endothelial lining is usually intact, but may later become ulcerated. The process never affects the whole circumference of the vessel. Opposite these atheromatous patches the muscular lamina may be atrophied, but in the other parts of the circumference of the vessel it may actually be in a condition of hypertrophy.

As regards the diffuse variety, or as it is better termed merely "arteriosclerosis", much diversity of opinion exists.

Dr. Mott agrees with Thoma in the following description of diffuse arteriosclerosis. He says that the condition begins in the small arteries of the kidney, brain, and heart, as a hyaline swelling of the muscular fibres of the media, followed by fatty degeneration and atrophic changes. He then indicates that the intima is thickened and represented only by a homogeneous hyaline material/

material with but few nuclei. He goes on to state that the results of these widespread changes are, increased resistance to the flow of blood through the capillaries, hypertrophy of the left ventricle, dilatation of the larger arteries from degenerating changes in the muscular and elastic tissues of the media, slowing of the circulation and compensatory proliferation of the subendothelial layer of the inner coat.

Sir George Johnson ascribed thickening of the arteries in kidney disease to a hypertrophy of the muscular coat; Gull and Sutton to a fibriotic change in the arteries and capillaries, beginning usually but not always in the kidney, which they term "arteriocapillary fibrosis."

Sir Wm. Broadbent admits that the muscular coat of the arteries is increased; while Lanceraux defines the condition as a proliferation of the cells of the intima which goes on to fatty degeneration and a failure of nutrition.

Rosenstein agreed with Johnson as to the thickening of the muscular coat of the arteries but thought that the thickening of the intima was inflammatory.

Ewald held that there were two forms of changes in the arteries in Bright's disease; first that in which the kidney was affected primarily and was followed by hypertrophy of the heart and muscular coat of the arteries, and secondly that in which the disease of the general/

general vascular system was an arteriocapillary fibrosis and was the starting point of the kidney disease.

Savill, in 1897, described a condition of the arteries in which a hypertrophy of the muscular coat was present and which he called "Hypermyotrophy."

The above may be taken as examples of the two schools which existed a few years ago, one of which held that the muscular coat was hypertrophied in arteriosclerosis; the other that it was not, and that the chief change occurred in the tunica intima with a degeneration of the tunica media.

Russell[1], in 1907, reconciled these two schools of thought by stating that the confusion which existed in this subject was due to the fact that some observers applied the changes found in the arteries inside the kidney to all the arteries of the body. He stated that the changes found in the peripheral arteries of the body were (1) a marked thickening of the tunica media due to a hypertrophy of its muscular fibres; (2) a thickening of the tunica intima due to hyperplasia of its subendothelial connective tissue without atheromatous change; (3) in some instances fibrous hyperplasia and thickening of the tunica adventitia. He said that the muscular coat might show some degeneration but that the prevalent notion that the muscular coat was the seat of fibroid degeneration was entirely erroneous. He further stated that these changes were not confined to limited areas of the vessel wall as in atheroma, but affected/

affected uniformly the whole length of the artery. He described in the same cases, in addition to these changes in the principal arteries, a condition of the internal arteries of the kidneys, showing a marked hyperplasia of the tunica intima while the tunica media was atrophied. He laid particular stress on the fact that while the internal arteries of the kidney undoubtedly exhibited the changes described by Thoma and Mott, i.e., hyperlasia of the intima with atrophy of the media, the peripheral arteries showed a condition of hypertrophy of the media with thickening of the tunica intima but without atheromatous change. He applied the term "arteriosclerosis" clinically to all thickened vessels other than those thickened by atheromatous degeneration, and included (a) pure hypermyotrophy, (b) hypermyotrophy with thickening of the internal coat, and (c) those in which the adventitia was also thickened.

While I have not examined arteriosclerotic arteries microscopically to any great extent, my clinical experience has led me to the opinion that, whatever other condition may be present in the thickened arteries which one sees in hospital, in the great majority the muscular coat is undoubtedly not degenerated, but that on the contrary it is either hypertrophied or in a condition which renders it especially sensitive to external influences; and moreover that this coat is liable to contract and relax to an extent markedly in excess of the normal. I base my opinion, as I will endeavour to show/

show by means of a series of cases, on the marked beneficial results which can be obtained by overcoming this tendency of the muscular coat to go into a condition of hypertonic contraction.

Other varieties of thickened arteries which may be mentioned in passing are:-

(1) Those showing a condition of endarteritis obliterans in which the change is in the intima but is more or less diffuse, and which may or may not be due to syphilis.

(2) Those which show a calcareous degeneration of the media.

In addition to the above types, the walls of arteries may undoubtedly be thickened by a condition of hypertonic contraction or spasm of the muscular fibres of the media, with, or without, a condition of arteriosclerosis being present.

Before proceeding further with this subject, it is essential that we should have a clear understanding in our minds as to what is the effect, of thickened and narrow arteries generally on the blood pressure. It is a well-known clinical fact that thickened arteries do give rise to a high Sphygmomanometer reading, which means that an increased pressure is necessary to occlude the brachial artery by means of a pneumatic bag encircling the upper arm. This signifies that an increased resistance is present, and we naturally ask ourselves, What/

"What is this resistance due to; is it simply due to an increased pressure of the blood inside the artery, or is some additional factor present?" The only factors which could influence the reading, apart from actual blood pressure, are (a) the vessel wall and (b) the soft tissues surrounding the artery. The latter of these can be excluded as it has been quite definitely shown by Janeway[2] that the soft tissues do not influence the reading to any great extent, and I have often satisfied myself that the reading obtained from an arm is the same, whether the armlet is applied above or below the clothing. The question as to the effect which the vessel wall has upon the Sphygmomanometer reading is one which has given rise to considerable controversy. Some authorities hold that the difficulty in overcoming the resistance of the vessel wall, irrespective of the pressure of the blood inside, is the chief cause of the high readings obtained in cases of arteriosclerosis, while others are equally convinced that such is not the case.

The question comes to be, are the readings given by the Sphygmomanometer a true measure of the pressure of the blood inside the arteries, or does the resistance of the arterial wall play an important part?

Von Basch,[3] in 1887, found that the pressure necessary to close the empty radial artery was 1 mm. of Hg. for a normal vessel and 7 mm. of Hg. for a sclerosed one.

Martin,[4] in 1905, in a few experiments on the normal carotids/

carotids of man, horse, and dog, found that a pressure of 2 mm. was sufficient to collapse the arterial wall, in arteries from an advanced case of arteriosclerosis only 7 mm. were required.

Herringham and Womack,[5] in 1908, examined carotid, iliac, and brachial arteries, removed post-mortem from 49 bodies. The results showed wide variations, the extremes in the case of the brachials being 4 to 34 mm., these variations having no relation to the age of the patient or the condition of the vessel wall.

Scholtyssek,[6] in 1909, performed a series of experiments on the resistance of a living carotid of a rabbit. His results showed a resistance of 2 mm. for a rabbit's carotid, and 7, 8, 11, and 15 mm. for a dog's similarily tested.

Schmidt,[7] in 1909, described a number of observations on freshly removed dog and rabbit arteries. He tested the effect of varying internal pressure, of temperature, of painting with epinephrin and with chloral hydrate, and of death of the artery. With large arteries from these animals he found the resistance to range between 1.84 and 7.38 mm. of Hg. He was unable to produce noticeable constriction by painting with epinephrin or dilatation by chloral hydrate.

Professor W. Russell,[1] in 1908, was the first exponent of the view that the high Sphygmomanometer readings obtained in cases of arteriosclerosis are not due to true/

true blood pressure. He agrees with Huchard in laying considerable stress on the occurrence of Hypertonus of the vessel wall, in both normal arteries and those with hypertrophical muscular coats. This Hypertonus rather than arterial hypertension is what he believes to be measured. In his book on the subject he gives numerous clinical cases and experiments in support of his contension.

Williamson,[8] in 1909, published a paper in which he comes to the conclusion that resistance due to the arterial wall may markedly influence the readings, his reasons for this statement being:-

(1) In cases of definitely high arterial blood pressure the systolic reading obtained from the legs is in nearly all cases markedly higher than that obtained from the arm of the same patient, the diastolic readings being identical.

(2) Such a difference between the systolic arm and leg readings is the exception in cases of normal or low blood pressure without obvious arterial thickening.

He attributes this difference to the fact that the arteries of the leg are more affected than those of the arm in cases of arteriosclerosis.

Professor Leonard Hill,[9] in 1909, in his discussion of Williamson's paper, states that his own investigations of the blood pressure in the arm and leg failed to show any appreciable difference in the readings, if certain precautions were taken, except in the case of free aortic regurgitation. Four years later Russell Wells and Leonard Hill[10] published a paper in which they admit that the readings obtained in the leg are often higher than those obtained in the arm of the same patient/

patient, and go on to say that these readings can be diminished by causing vaso-dilatation in the leg arteries; they, therefore, conclude that this higher leg reading is due to the fact that the arteries are in a state of contraction. They explain this phenomenon by the statement that the pulse wave is conducted along a rigid tube with less loss of power than along an elastic one. They support this contention by a series of experiments with rubber tubes of varying thickness of wall and elasticity.

Janeway[11] was previously of the opinion that the vessel wall offered no resistance to compression, but in 1910, in conjunction with Park,[12] he published a paper in which he comes to the following conclusions:-

(1) The arterial wall offers definite resistance to compression but this is practically negligible with normal arteries and a normal range of blood pressure.

(2) Small arteries with thin walls are more readily compressed than large arteries with thick walls.

(3) Atheroma does not increase the resistance and calcification only to a small degree, if segments longer than 6 cm. are examined.

(4) The only factor determining the compressibility of an artery, which seems capable of introducing an error of real importance in the clinical measurement of systolic blood pressure, is the state of contraction of its walls. A degree of hypertonic contraction of the brachial artery sufficient to cause an error of more than 30 mm. of Hg. seems improbable, and of more than 60 mm. incredible, during life.

He bases these conclusions on the results of experiments conducted on the arteries of various animals, including human beings, which had been removed from the body/

body and which were examined under the influence of various physical and chemical agents, i.e., intense cold, warmth, epinephrin and Barium Chloride.

Sherrington and McKenzie[13] state that there is still some uncertainity whether the arterial wall itself may not oppose some resistance to compression, either from changes in the wall or from its degree of contraction. They go on to say that an important point brought out by the measurement of arterial pressure is that what is commonly spoken of as heart failure is not necessarily accompanied by a fall of arterial pressure.

In many cases of heart failure with great breathlessness on exertion it will be found that the blood pressure has not fallen, and when recovery takes place the blood pressure has not risen.

We are thus faced with two schools of thought on this subject, one of which says that the vessel wall does not oppose any, or only slight, resistance to compression, while the other says that the arterial wall opposes a very considerable resistance. The question is "Which are we to believe?"

To my mind the greatest point of weakness in the arguments of the first school is the fact that the majority of them found their contentions on results obtained from experiments on arteries removed from the body either before or after death. Von Basch, Martin, Herringham and Womack, and Schmidt were all working on arteries removed post-morten. This fact, I think, renders/

renders their work of little value when applied to living arteries.

Leonard Hill[9] for years held to the opinion that the arterial wall was a negligible factor; finally, however, he admits the difference in Sphygmomanometer readings obtained from the arm and leg, but he attributes this well-known clinical phenomenon to the better conductivity of the pulse wave along sclerosed arteries as opposed to normal ones. One must admit that in an elastic artery a certain amount of the force of systole is expended in distending the vessel wall, the elasticity of this wall being then brought into play to keep up the diastolic pressure. We have, however, an undoubted clinical phenomenon which we cannot explain if we accept Hill's theory. I refer to the fact of the Sphygmomanometer reading obtained from the fore-arm being in many cases higher than that obtained from the upper arm. This is a condition which I have many times satisfied myself was present, and in the 21 cases of high blood pressure which Williamson quotes in his paper on the subject, 9 have a higher systolic pressure in the fore-arm than in the upper arm. If Hill's view as to the better conductivity of the pulse wave along a rigid artery were the only explanation of high pressure, we could not explain this phenomenon; if however, we accept the theory that the arterial wall itself opposes a resistance to compression, the phenomenon is quite simple of explanation. No doubt the more rigid/

rigid an artery is the better is the pulse wave transmitted along it, but I do not believe that this entirely explains the high Sphygmomanometer readings which we meet with clinically.

Turning to the other school of thought, we find that its first exponent was Professor Wm. Russell.[1] He insists on the great importance of a thickened arterial wall and more especially of hypertonus of the muscular coat, be it hypertrophied or normal, in introducing a very considerable degree of error into the clinical estimation of blood pressure by the Sphygmomanometer. This view is also held by Williamson, and both these observers support their contentions by numerous clinical cases.

Janeway,[11] who at one time adhered to the view that the arterial wall was negligible, now agrees[12] that the vessel wall offers definite resistance to compression, especially when in a state of hypertonus, but thinks that Russell goes too far and attaches too high a value to this element of error in the estimation of blood pressure by the Sphygmomanometer. Janeway, however, was working with arteries which had been removed from the body, and though they still contracted under the influence of epinephrin and intense cold, yet I think that one is justified in assuming that they did not contract to the same extent as, under certain influences, they are able to do in the living body. In addition, much of his work was done on arteries from animals, and they probably had not hypertrophied muscular coats, which is doubtless an important factor influencing the amount

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of contraction which occurs.

Let us now look at the case from a purely clinical point of view. We have one fact which no one disputes, namely, that in cases with thickened and contracted arteries we do get a high Sphygmomanometer reading. Still keeping to the region of facts, we can now say without fear of contradiction, that in many cases the reading obtained from the legs is considerably higher than that obtained from the arms. From this we may draw the conclusion that, as we have acquitted the soft parts covering the arteries of any participation in increasing the reading, we must look for the reason in the vessel wall. Pathology and experimental evidence tell us that some thickening or rigidity of the vessel wall is present. Thus far all are agreed, but here we come to a parting of the ways. One school would have us believe that a thickened and rigid vessel wall produces a high Sphygmomanometer reading by allowing the pulse wave to be transmitted along the artery with more force, while the other school holds that the high reading is due to the resistance to compression of the vessel wall. In other words, one school says "We have here a true measure of the force of the pulse wave within the vessel;" while the other says "Here we are measuring in addition to blood pressure, the resistance of the vessel wall." Which are we to believe?

Some months ago I had under my care an old woman over 80 years of age. When I first saw her, she was suffering/

suffering from gangrene of the toes on both feet. The Sphygmomanometer readings obtained from the legs were considerably higher than those obtained from the arms. The posterior tibial arteries, felt behind the internal malleoli, were markedly thickened and resistant to the touch. Obviously the force of the heart was not sufficient to keep up the circulation in the lower limbs, and yet the Sphygmomanometer reading was high. After death, which occurred from an intercurrent pneumonia, I had the opportunity of dissecting out the main arteries of the body, and found the following conditions present. The arch of the aorta and the abdominal aorta down to the bifurcation were comparatively healthy. The brachials, radials, coronaries, and cerebral arteries were also free from any calcareous degeneration, while the iliacs, femorals, and tibials showed an advanced condition of arteriosclerosis, this accounting for the high readings obtained.

If we accept the teaching that the pulse wave is better transmitted along rigid arteries, how can we explain such a case as the one just quoted where the high pressure in the legs was associated with gangrene of the toes?

If Leonard Hill's explanation be the correct one, the force of the pulse wave which he poetically describes as "hammering the blood through the capillaries" should be sufficient to prevent gangrene. In this case, the pressure as given by the Sphygmomanometer was higher in the legs than in the arms, and yet I am convinced/

convinced that the actual blood pressure was no higher, if as high. I think that in this case the high reading was undoubtedly to a large extent due to the resistance of the vessel wall.

There was no doubt considerable obstruction to the passage of the blood through the capillaries, but if the systolic blood pressure had been as high as the Sphygmomanometer led us to believe, I do not think gangrene would have occurred, provided the arterioles were not entirely blocked - as was not the case.

In cases where the systolic reading in the legs is higher than that in the arms, as a rule the diastolic reading is slighter lower, but the much greater increase in the systolic reading would more than counteract the small diastolic diminution, if the high systolic reading were merely due to a better conduction of the pulse wave. While Leonard Hill's contention is no doubt true to a certain extent, I do not think that it altogether explains the high Sphygmomanometer readings, which I consider chiefly due to the resistance of the vessel wall.

In a subject such as this it is quite impossible to lay down rules as to what happens in life, from information gained "post-mortem," valuable as that may be. In dealing with the living body, we msut bear in mind a factor, which we are prone to forget, namely, that we are dealing with a living organism possessed of vital energy. This factor, unless remembered, is very apt

to/

to nullify any conclusions we may come to from the investigation of various phenomena after death. In the subject under discussion, this factor is especially important. That a vessel wall offers very little resistance to compression after death is no proof at all that it does not offer resistance during life. The only way to obtain trustworthy information is to examine the arteries carefully during life, comparing the information which we obtain by means of the Sphygmomanometer with that which our eyes, fingers, and ears convey to us. I have seen many cases in which the Sphygmomanometer persistently recorded a pressure of 200 mm. of Hg. or over, and yet my fingers conveyed to me the impression that the pulse was weak and the actual pressure of the blood low, and, moreover, from an examination of the general condition of the patient, the fact that the circulation was in a very poor condition was obvious.

With this information before me, I was quite unable for a moment to believe that the Sphygmomanometer gave me a true measure of the patient's blood pressure. It was inconceivable to suppose that a patient who had a dilated heart, breathlessness on the slightest exertion, and oedema of the feet and ankles, could have an actual blood pressure of 200 mm. of Hg. in the peripheral arteries.

From my own observations, therefore, I am convinced that the arterial wall does oppose a very marked resistance to the occulsion of an artery by external pressure, such/

such as the pneumatic armlet of the Riva-Rocci Sphygmomanometer.

I will at this point give three cases which show this point clearly.

Case I. Male, aged 57, suffering from Chronic Myocarditis, admitted to hospital complaining of general weakness. Eighteen months previous, he began to be troubled with excessive weakness and breathlessness. This had continued up till time of admission. For the three weeks previous he had been confined to bed. He had syphilis at the age of 23. His habits were somewhat alcoholic.

On admission to hospital, his pulse was 80 per minute, his respirations were 20, and his temperature 97°F. His pulse was small, his radial and brachial arteries were considerably thickened. The Sphygmomanometer reading taken from his upper arm was 200 mm. of Hg. Heart. No pulsation was visible. The apex beat was felt with difficulty in the fifth interspace just internal to the nipple line.

The right border of his heart was found to extend half-an-inch to the right edge of the sternum.

The left border was situated, in the fourth space, three and a half inches from mid-sternum, and in the fifth space in the nipple line, five inches from mid-sternum.

The heart sounds were very faint and somewhat irregular, but were closed in all areas. His urine was acid,/

acid, specific gravity 1015, contained a trace of sugar, but no albumen.

Progress and Treatment. He was admitted to hospital on April 3rd., 1913. His Sphygmomanometer reading was then 200 mm. of Hg. He had considerable dyspnoea. He was given Pot. Iod., grs. x., T.I.D., and Erythrol Tetranitrate gr. ¼ at nights.

On April 14th his vascular pressure was down to 185 mm. of Hg., and he was feeling much more comfortable. He was noticed to have a Pulsus Alterans.

On April 26th his vascular pressure was 180 mm. of Hg.

On May 8th his symptoms were so much improved that he was dismissed.

On January 14th., 1914, he was readmitted. He was cyanosed, very breathless even when lying in bed, and his feet were oedematous.

His pulse was slightly irregular and of small volume. His radial and brachial arteries were considerably thickened, and his vascular pressure was 175 mm. of Hg. His heart was considerably more dilated than upon the first occasion. The apex was situated in the fifth interspace, 6¼ inches from the mid-sternal line and 1 inch external to the nipple line. The right border extended to 2 inches from mid-sternal line. The sounds were feeble but closed.

His urine was acid, specific gravity 1070, contained albumen and was small in amount.

He/

He was very restless, and was given Digitalis, Pot. Iod., and Theocin Sodium Acetate.

On January 20th his oedema was somewhat decreased and he was passing 50 ozs. of urine. His vascular pressure remained the same. The Theocin was stopped.

On January 29th his vascular pressure had risen to 192 mm. of Hg. He was very noisy at nights and his condition showed practically no improvement. He was given 1 gr. of Erythrol Tetranitrate at night which quietened him.

On February 6th his vascular pressure was down to 175 mm. of Hg., and his general condition was somewhat better.

He was discharged on February 9th considerably improved.

Case II. Male, aged 68, suffering from Myocarditis with mitral incompetence.

He was admitted on February 22nd., 1913, complaining of marked dyspnoea and oedema.

His heart was considerably dilated, the sounds were weak and a mitral systolic murmur was heard.

His arteries were considerably thickened and tortuous, and his vascular pressure was 210 mm. of Hg.

There was considerable oedema at the bases of his lungs.

He was given Ammonia Senega mixture, Tincture of Squills, and Erythrol Tetranitrate gr. 1 at night.

On/

On March 6th his vascular pressure was 166 mm. of Hg. His breathlessness and oedema were considerably diminished and his general condition showed a marked improvement.

On March 13th his vascular pressure was 172 mm. of Hg. and the improvement continued.

On April 4th he was feeling quite easy and his breathlessness was much better though still somewhat short.

He was dismissed on April 16th feeling much better.

Case III. Male aged 68. Admitted complaining of attacks of breathlessness. He had two of these attacks, one one week and the other three weeks before admission.

He had no venereal history, and was a moderate drinker.

On admission he looked cyanosed and was very short of breath.

His pulse was regular, rate 76 per minute, and was fairly well sustained. The vessel wall was thickened and tortuous and the vascular pressure was 198 mm. of Hg.

The heart was enlarged, the sounds were faint, and in the mitral area a systolic murmur was heard.

He had a large thin-walled aortic aneurism. He had some bronchitis and oedema of the bases.

His urine was acid, specific gravity 1010, and contained a trace of albumen.

Three days after admission, he suddenly became very blue and cold, and extremely breathless. His arteries/

arteries seemed to be tightened up and were very resistant to the touch. His vascular pressure was higher than before. He was given Digitaline, gr. 1/100, and a vasodilator. Concomitant with the relaxation of his arteries, his breathlessness diminished and he was much easier. This attack lasted about half-an-hour.

The day following, he was put on Erythrol Tetranitrate, gr. ss., twice a day. Next day he had another breathless attack similar to the first, but not so severe. He was given the same remedies. From this time onwards he had no more breathless attacks. His vascular pressure continually remained about 200 mm. of Hg. One month after admission, he was allowed up and developed oedema of the feet and ankles. He was put back to bed for a few days and the oedema cleared up. At this time the vascular pressure was as follows:- Right arm, 202 mm. of Hg., Left arm, 205 mm. of Hg.

He remained in hospital one month longer, his vascular pressure remaining about 200 mm. of Hg. the whole time. He was then dismissed.

A fortnight later, he was readmitted having been seized with another severe attack of breathlessness. On admission, he had marked oedema of the feet and ankles. His pulse was barely perceptible and was beating at 120 per minute. His breathing was wheezy, but the acute attack had been relieved by means of Liq. Trinitrini given to him by the doctor who was called to see him. The walls of his brachial and radial arteries were greatly thickened and tortuous. The vascular pressure/

pressure was 190 mm. of Hg.

His heart was considerably more dilated than on the previous occasion and the sounds were very feeble. The mitral systolic murmur was distinctly heard. His lungs were oedematous. Under similar treatment, i.e., cardiac tonic and vaso-dilator, he showed a marked improvement as on the previous occasion. His vascular pressure never fell below 190 mm. of Hg.

These three cases all show the same phenomenon, namely, a high Sphygmomanometer reading along with the signs and symptoms of a failing heart. They also bring out the fact that as the heart obviously increased in strength, the blood pressure, if we take the Sphygmomanometer as an indication of blood pressure, not only did not show a corresponding rise but actually a fall. This fall which was comparatively small in amount occurred on the administration of a vaso-dilator, and synchronously with it there occurred the relief of symptoms.

The following case showed the same phenomenon but to a more marked degree.

Case IV. Male, aged 45, admitted to hospital complaining of cough and shortness of breath.

Two years before admission he suffered from a similar condition. He recovered and was working up till twelve weeks before admission, when he again developed cough and breathlessness and oedema. His condition gradually became worse, and lately he was considerably troubled with vomiting and loss of appetite.

He/

He gave no venereal history, but was a heavy drinker and smoker.

On examination his pulse was 76 per minute, his respirations 28, and his temperature 98°F.

He was very breathless on the slightest exertion, he was cyanosed, and his legs and feet were very oedematous.

His pulse was regular and moderately well sustained. His brachial and radial arteries were thickened and resistant to the touch. The brachials gave a Sphygmomanometer reading of 194 mm. of Hg. The veins in the neck were full and pulsating. On palpation of the precordia the apex beat was diffuse, best felt in the sixth interspace one inch external to the nipple line. A systolic thrill was felt over the second right interspace.

On percussion of the heart, the right border was found to extend one-and-a-half inches from the mid-sternal line, and the left border, in the fifth interspace five inches, and in the sixth interspace five and a half inches from the same place. On auscultation a systolic murmur was heard in all areas, the points of maximum intensity being at the aortic and mitral areas; the murmurs in these two areas being apparently of different origin.

Crepitations were heard over both bases, and the liver was somewhat enlarged.

The kidneys were not palpable, but the loins were slightly tender.

The/

The urine was acid, specific gravity 1012, and contained albumen and blood. Microscopically it showed red blood corpuscles, hyaline and granular casts, and renal epithelium.

Progress and Treatment. On July 9th (the day after admission) he was very breathless and oedematous, he was coughing a good deal. Passed 18 ozs. of urine, Sphygmomanometer reading, 196 mm. of Hg. He was given Ammonium Carbonate and Tincture of Squills.

On July 19th he showed no improvement, in fact, he was decidedly worse. He was very pale and short of breath, and had developed dullness and feebleness of breath sounds at the base of his right lung. His vascular pressure was 200 mm. of Hg. His pulse was 106 per minute, respirations 38, and temperature 98°F. He only passed 10 ozs. of urine on the previous day.

He was given Digitaline, gr. 1/240, and Erythrol Tetranitrate, gr. j., twice a day.

On July 21st he was much improved, his oedema was less, and his breathing much better. He passed 22 ozs. of urine as compared with 10 ozs. two days before. His vascular pressure was 154 mm. of Hg. His pulse was 100 per minute, his respirations 28, and his temperature 98°F. On the following day his condition still showed improvement. His vascular pressure was 162 mm. of Hg., his pulse 78 per minute, his respirations 28, and his temperature 97·6°F.

On July 25th his oedema was more marked. Ten ounces of fluid were removed from his chest/

chest and Southey's tubes introduced into his legs. He passed 30 ozs. of urine, Pulse 64, Respirations 24.

On July 28th he was somewhat better, and passed 40 ozs. of urine.

On July 31st the improvement was continuing.

On August 1st he became suddenly worse about 5 a.m. and died at 11.30 a.m.

The Post-Mortem examination showed the heart to be greatly enlarged, all four cavities being extremely dilated. A condition of aortic obstruction with mitral regurgitation was present.

The coronary arteries showed a slight fatty change only.

The aorta was practically healthy, except for the presence of a few patches of early atheroma in the arch.

This case, on admission, showed a Sphygmomanometer reading of almost 200 mm. of Hg.; in addition to this, the circulation was very weak as evidenced by the faint heart sounds, oedema, cough, and breathlessness which were present. Ten days later, the patient's condition was worse. His circulation was more embarrassed, as shown by the augmentation of all his previous symptoms and by the fact that he was passing only 10 ozs. of urine per diem, and yet, in spite of all this, the Sphygmomanometer still registered a pressure of 200 mm. of Hg. The point that impressed itself on my mind was the fact that, although the circulation, and therefore the power of/

of the heart, was weaker than on admission, yet the pressure as given by the Sphygmomanometer was not only not diminished but was actually increased.

At this stage a vaso-dilator was given, and the patient showed an immediate improvement, his symptoms, as shown by the progress notes taken each day, were markedly relieved. Now, instead of finding, as we should expect, a corresponding increase of the blood pressure, presuming that the Sphygmomanometer gives us a true measure of blood pressure, we find on the contrary a fall of something like 60 mm. of Hg. In the face of these facts, the obvious conclusion to be drawn, I think, was that the Sphygmomanometer did not give a true indication of the patient's blood pressure; what the reading did give was, in addition to the blood pressure, a measure of the resistance of the vessel wall. My reason for making this statement is that after the administration of a vaso-dilator, the vessel wall felt distinctly less resistant, and yet at the same time the strength of the pulse wave, as determined by the finger, felt decidedly greater and, in addition to this, the symptoms of cardiac deficiency were greatly relieved. Unfortunately the relief was only temporary, as the organic condition of the man's heart precluded any permanent improvement from any therapeutic agent.

This case also brings out another point, which I will go into more fully later, namely, that although the man's arteries showed a considerable amount of organic change, yet/

yet the resistance and thickening of their walls was to a certain extent due to a condition of spasm of their muscular coats, which was relieved by a vaso-dilator; this being in itself a proof that in arteriosclerosis the muscular coat is not always degenerated. The effect of this vaso-dilator was, I take it, by relieving the spasm, to increase the lumen of the arteries, and also as a necessary result, to diminish the thickness of their walls, and therefore their resistance. The effect of this on the heart would be, by diminishing the peripheral resistance to the blood flow, to enable it to cope more successfully with the work before it; and the actual strength of the heart would therefore be increased, thus involving a rise in the true blood pressure.

Physics teaches us that if in any system, through which there is a steady flow of fluid, a wide-spread constriction occurs in any part, the pressure of the contained fluid in the constricted tube diminishes, whilst the pressure proximal to the constriction rises. When this occurs in the circulatory system, we have the following sequence of events. The peripheral arteries contract and therefore their lumen is diminished; as a result, the aortic pressure rises while the pressure in the contracted arteries falls. In addition to this, the total capacity of the arterial system is diminished, and, as the total quantity of blood remains the same, that which is driven out of the arterial system must be accommodated in the venous system which therefore dilates. Meanwhile, the pressure in the small arterioles and capillar-

ies/

capillaries is lower than normal, and the heart, in order to keep this up to the required standard, beats more forcibly and forces the blood through the contracted arteries more quickly. In order to do this, the heart calls forth its reserve power and, if the heart is healthy, for a time all goes well, and no symptoms occur. Eventually, however, if the heart is not quite healthy, it becomes exhausted, and we get backward pressure into the pulmonary and venous circulations and cardiac dilatation, and, following this, we get the whole series of symptoms and physical signs which we call cardiac insufficiency.

By this time the heart is rapidly becoming weaker, and eventually may not be beating with its normal force; this is evidenced by weakness of the heart sounds. All this time it must be borne in mind that while the aortic pressure is high, the pressure in the contracted arteries and the region beyond is lower than normal.

When we have, as in the cases quoted, in addition to these signs and symptoms of heart failure, a Sphygmomanometer reading of 200 mm. of Hg. in the contracted arteries, we must come to the conclusion that this is not true blood-pressure. We know that the reading is obtained from the contracted arteries because we can feel these arteries relax on the administration of a vaso-dilator.

Another point is that we have a high pressure in the brachial arteries; if this was entirely due to peripheral/

peripheral resistance we should expect to find that the pressure would be lower in the radials, and yet such is not by any means always the case, the Sphygmomanometer reading may, in fact, be higher in the fore-arm than in the upper arm.

I think that looking at the question from a clinical standpoint, the evidence indicates quite definitely that the reading which we obtain, by means of the Sphygmomanometer, from contracted and thickened arteries, for as I have shown, the arteries are not only thickened but also contracted, is not a true measure of the blood pressure, which in these cases is undoubtedly lower than normal, but that it is, in addition, a measure of the resistance opposed to compression by the vessel wall. Granting that contraction of the wall does occur, our object must be to overcome this contraction, and by so doing diminish the peripheral resistance, and thereby relieve the embarrassed circulation. As shown by the previous cases, and also to a more marked extent by those following, the administration of a vaso-dilator attains this object.

Not only do we have this contraction occurring with a moderate amount of muscular coat remaining, but we may have it, as thefollowing cases will show, to a much more marked degree in either a muscular coat of normal thickness, or in one which is greatly thickened or hypertrophied. In these cases, we may have the vascular pressure, as opposed to the true blood pressure, falling to the extent of 50 or even 100 mm. of Hg. on the administration of/

of a vaso-dilator, and along with this the signs of cardiac failure, which have before been very prominent, disappearing. I will now quote a number of cases which show these phenomena.

Case V. Male, aged 40, suffering from Aortic disease. For some time before admission he had suffered from anginal and breathless attacks, which he stated had been getting worse and more frequent. He gave a history of syphilis.

On admission, he was very breathless and was markedly cyanotic. He had a cough and complained of a feeling of choking. He was unable to lie down on account of his dyspnoea, and looked anxious and uncomfortable.

His radial pulse was rapid and irregular in time and force, the rise and fall was sudden. The vessel wall showed some tortuosity and regular thickening. Vascular pressure, 165 mm. of Hg.

His heart was enlarged, the apex beat was in the sixth interspace five inches from the mid-sternal line. On auscultation a double aortic murmur was heard together with a mitral systolic.

His other systems were normal.

His urine was acid, specific gravity 1020, and contained no abnormal constituents.

Progress and Treatment. On January 25th (day of admission) his vascular pressure was 165 mm. of Hg., his pulse was 104 per minute, and his respirations 24.

He was given a cardiac tonic

and/

and diuretic.

On January 29th, his condition showed a distinct improvement, his vascular pressure was 140 mm. of Hg., his pulse 70 per minute, and his respirations 24.

On February 14th his vascular pressure was 175 mm. of Hg., and, although he had several anginal attacks, his general condition was greatly improved.

On February 27th his vascular pressure was 132 mm. of Hg.

On March 14th his vascular pressure was 182 mm. of Hg.

He was discharged, all signs of cardiac distress having disappeared, but his anginal attacks were never quite cured. During the whole period of his residence in hospital, the Sphygmomanometer readings obtained from his brachials varied to a considerable extent as will be seen from the instances quoted.

Comment. This case shows a remarkable variability of vascular pressure without any vaso-dilator having been administered. At one period his pressure was as high as 182 mm. of Hg., and at another as low as 132 mm. of Hg. It is difficult to suppose that the power of his heart varied between these wide limits, and so we are driven to look for the cause in the vessel wall. A wall which was organically thickened, by which I mean one that was in a condition of calcareous or fibrous degeneration and devoid of muscular substance, would not have shown any variation. The only explanation which I can see is that the/

the muscular coat was present, and was alternately in a condition of hypertonus and hypotonus.

A more marked and lasting result will be seen in the two following cases, in neither of which was a vaso-dilator given, the only treatment being rest in bed; and yet in both cases the relief of the cardiac symptoms was accompanied by a fall in vascular pressure.

Case VI. Male, aged 36, admitted with a history of pain in left lumbar and iliac regions, severe frontal headaches and breathlessness. The headaches came on suddenly, were situated in the frontal region, lasted for a few minutes, and then gradually passed off. He had on an average ten of these attacks of pain in the 24 hours. He had no venereal history.

He was breathless on the least exertion and took momentary faint turns. He was somewhat flushed and slightly inclined to be cyanotic.

His pulse was 72 per minute, moderate rise and fall, and the apex was fairly well sustained. The vessel walls felt slightly more thickened and firm than one would expect in a man of his age. His vascular pressure was 158 mm. of Hg.

His heart was not markedly enlarged, the sounds were somewhat faint, and a soft blowing systolic murmur was present in the mitral area.

His breathing was distinctly embarrassed when lying down, and he had a morning cough.

His/

His kidneys were somewhat tender on pressure, chiefly the left.

His urine was acid, specific gravity 1008, and contained no albumen but a large number of streptococci.

Chromo-cystoscopy showed nothing abnormal.

His other systems were normal.

He was in hospital for one month, but received no medicinal treatment for his cardiac condition.

During his stay his cardiac symptoms quite disappeared, and his vascular pressure fell steadily from 160 mm. of Hg. to 120 mm. of Hg., occasionally falling as low as 110 mm. of Hg. Along with this fall in pressure, his vessel walls became soft and relaxed. As his pressure fell, so the symptoms of an embarrassed heart cleared up, and on his discharge, his heart was beating much more powerfully than on admission as measured by the strength of the sounds and the feel of the pulse, although the Sphygmomanometer gave a much lower reading.

Case VII. Male, aged 38. Suffering from Pulmonary Stenosis with loss of compensation.

Admitted to hospital complaining of cough and shortness of breath. A few days before admission, he suddenly became very breathless on climbing a hill. He developed a cough, and the breathlessness continued even when lying in bed. A year ago he had a similar attack and was laid up for six months with it.

On examination he was suffering from severe palpitation and breathlessness; he had to be propped up in/

in bed by three pillows. His pulse was flickering, it had a small excursion, the rise was gradual and the fall sudden. His pulse rate was 130 per minute, and his respirations 28 per minute. On inspection of the precordia, the apex beat was scarcely visible, in fact the only visible pulsation was in the epigastrium. On palpation, a faint pulsation was felt over the whole precordia.

On percussion, the right border of the heart was situated one inch to the right of the mid-line of the sternum. The apex was situated in the sixth interspace, one inch below and one and a half inches external to the left nipple.

The heart sounds were soft and indistinct. A long, blowing, soft murmur was heard during ventricular systole, faintest in the mitral and tricuspid areas, increasing in intensity as one approached the aortic area, and loudest of all in the pulmonary area.

Ronchi were heard all over the chest, and some crepitations were heard at both bases. The percussion note was resonant.

The urine was acid, specific gravity 1020, and contained no abnormal constituents.

Table/

	Pulse Rate.	Sphygmomanometer Reading.	Urine.
On day of admission	130 per min.	162 mm. of Hg.	16 ozs.
" second day	112 " "	156 " " "	22 "
" fourth day	104 " "	140 " " "	32 "
" sixth day	100 " "	150 " " "	38 "
" tenth day	98 " "	148 " " "	46 "
" thirteenth day .	Heart beating much more strongly, and oedema at bases considerably diminished.		
" fifteenth day .	104 per min.	146 mm. of Hg.	46 ozs.
" sixteenth day .	96 " "	128 " " "	48 "
" seventeenth day.	92 " "	124 " " "	42 "
" twentieth day ..	93 " "	128 " " "	32 "
" twenty-sixth day	92 " "	122 " " "	48 "
	Pulse is of good volume and steady, the breathlessness and cough are practically gone.		
" forty-second day	96 per min.	130 mm. of Hg.	65 ozs.
	Patient got up, and heart showed no symptoms of failure.		

These two cases are somewhat similar from the fact that both patients were young men; they both suffered from symptoms of cardiac failure; they both came into hospital with pressures of 160 mm. of Hg., and in both cases as the cardiac symptoms were relieved, the vascular pressures sank to normal, and, correspondingly, the arteries, which at the time of admission felt hard and resistant, became soft and normal to the touch.

I think that in these two cases, from the fact that the pressure sank to normal, we are justified in assuming that no organic thickening of the vessel wall was present, but that the hardness and the resistance of the arteries were due to a hypertonic contraction of the muscular coat, and that rest in bed and general treatment relaxed this hypertonus by presumably removing the influence of some irritant.

The/

The result of a hypertonic contraction of the muscular coat of a normal artery is to diminish its lumen, and also, as a natural result, to increase the thickness of its wall, as we have a smaller space than before to be surrounded by the same quantity of tissue. A second result of this condition is, as in the case of any muscle in a state of contraction, to increase the hardness and resistance to pressure of that muscle, which in this case is the wall of the vessel. We thus have a condition present which closely resembles arteriosclerosis, both in its physical phenomena and in the symptoms to which it gives rise; it differs from arteriosclerosis, however, in the fact that it is merely a transitory state and can be got rid of by appropriate treatment. It may, however, if allowed to go on for a sufficient length of time, as pointed out by Russell, give rise to an actual hypertrophy of the muscular coat and, eventually, to a conditon of true arteriosclerosis.

The results in these two cases would undoubtedly have been obtained more quickly if a vaso-dilator had been given.

The following is a case similar to the two previous ones.

Case VIII. Male, aged 65, admitted to hospital complaining of cough, palpitation, breathlessness and faintness, giddiness and anginal pains.

He gave a history of syphilis thirteen years before admission.

On examination, the heart was markedly dilated/

dilated, and the signs of cardiac insuffiency were obvious. There was marked pulsation over the precordia, the apex beat was diffuse, and situated in the sixth interspace one inch external to the nipple line. The apex was slightly external to this, and the right border extended to one inch beyond the right edge of the sternum. On auscultation a well-marked murmur of aortic regurgitation was heard. The pulse was irregular and of the water-hammer type. The vessel walls felt thickened and hard and the vascular pressure was 170 mm. of Hg. Both sides of the chest showed very marked signs of oedema of the bases of the lungs. The urine was acid, specific gravity 1020, and contained no abnormal constituents.

A week after admission, his vascular pressure had fallen to 158 mm. of Hg., and his general condition showed remarkable improvement. A week later his vascular pressure had fallen as low as 140 mm. of Hg., and his vessel walls were decidedly softer than on admission. Along with this, his symptoms were very much relieved. The oedema of his lungs had considerably diminished in amount, his dyspnoea was much better, and he was sleeping well at night.

After this his condition continued gradually to improve up till the time of his discharge from hospital. His vascular pressure remained comparatively low - varying between 135 and 145 mm. of Hg., and his symptoms remained in abeyance.

In this case, the pressure did not fall quite so/

so low as in the two previous cases. The patient, however, was a man aged 65, whereas the other two were much younger, so I think that we may take it that a pressure of 135-140 mm. of Hg. was in his case as low as we could hope to bring it, as he, no doubt, had a certain amount of organic thickening, probably hypertrophy, of the muscular coat which could not be removed.

The next case shows the more rapid effect which can be obtained by means of a vaso-dilator.

Case IX. Male, aged 67, admitted to hospital with a history of breathlessness on the slightest exertion and discomfort in epigastrium after food. This condition of affairs had been in existence for some nine months. He gave no venereal history, but had been a heavy smoker and a moderately heavy drinker.

On examination, his lips and ears were found cyanosed and he showed a considerable amount of dyspnoea.

His pulse was irregular, the expansion was slow and moderate in amount, and the apex was fairly well sustained. The vessel wall was irregularily thickened and felt distinctly hypertonic. The Sphygmomanometer gave a reading of 218 mm. of Hg.

The heart was dilated, the apex being situated in the 6th intercostal space, five and a half inches from the mid-sternal line. The right border was found to extend 2 inches from the mid-sternal line. On auscultation a soft blowing systolic murmur was heard

replacing/

replacing the first sound in the mitral area, and in the aortic area a short rough systolic murmur was heard. In the other areas both sounds were closed. Some signs of consolidation were found in the right infraclavicular region.

As regards the digestive system, he complained of discomfort in the epigastrium after meals, but never had any vomiting. Nothing abnormal was found on examination of the abdomen. His urine was acid, specific gravity 1026, and contained no abnormal constituents.

On admission, he was very breathless and cyanosed and had a pulse of 76 per minute, respirations of 20, a temperature 98°F., and a vascular pressure of 218 mm. of Hg. He was given ℥iij of Liquor Trinitrini and a dose of salts.

Next morning his symptoms were considerably relieved, and his vascular pressure was down to 146 mm. of Hg.

The change in the condition of his arteries at once arrested one's attention. The walls still felt slightly thickened, but the hardness and resistance so noticeable the night before had quite passed off.

Under further treatment by a vaso-dilator, his arteries remained soft and his vascular pressure never exceeded 150 mm. of Hg., usually being considerably lower, and along with this the symptoms of which he complained on admission quite disappeared. After being in hospital a month he was discharged, feeling as he expressed it, "a new man," and on the day of his departure the Sphygmomanometer/

Sphygmomanometer registered a pressure of only 132 mm. of Hg. This, compared with a pressure of 218 mm. of Hg. on admission, was certainly striking.

In this case, under a vaso-dilator, the patient's vascular pressure fell in a few hours from 218 mm. of Hg. to 146 mm. of Hg., and his cardiac symptoms were greatly relieved. This seems to me to be further undoubted evidence that the thickness and hardness of the vessel wall due to hypertonus, was the explanation of his high vascular pressure.

In this case, as in the previous one, there was a certain amount of actual muscular hypertrophy present in the walls of the vessels, and, in addition, as in the ~~two~~ previous case, the patient's actual blood pressure was probably slightly in excess of what is generally given as normal, though, considering the ages of the patients, quite normal for them. We frequently find cases, generally well advanced in years, in which we can bring down the vascular pressure, by diminishing the thickness and resistance of the vessel wall, to a certain point, but beyond that point no amount of nitrite will enable us to get.

The following is such a case -

Case X. Male, aged 64, admitted to hospital complaining of breathlessness and pain in the epigastrium. For some time before admission he had been troubled with cough and breathlessness, and he had noticed that his extremities/

extremities were often cold and blue. He was always troubled with constipation, and took a good deal of tobacco and alcohol.

On admission to hospital there was a considerable degree of dyspnoea and cyanosis present. The veins of the arms and legs were turgid and the extremities were cold. He complained of a considerable amount of precordial pain. He was unable to lie down, and he was restless and excited. His heart was enlarged and the apex beat was somewhat feeble but the sounds were closed in all areas. His pulse was irregular in time and force and the rise and fall were somewhat slow. The radial and brachial arteries were thickened and tortuous, the thickening seemed to be regular, and the vascular pressure was 200 mm. of Hg. The other systems were normal.

The urine was acid, specific gravity 1015, and showed a trace of albumen and hyaline and granular casts.

On September 6th (day of admission) his pulse was 90 per minute, his respirations 26, and his temperature 97·4°F. His vascular pressure was 200 mm. of Hg., and his arteries felt thickened and hard. He was given a cardiac tonic and a vaso-dilator.

On September 8th his vascular pressure was 185 mm. of Hg.

On September 9th his vascular pressure was 175 mm. of Hg. and his symptoms were considerably improved.

On September 24th his vascular pressure had fallen to/

to 140 mm. of Hg., and he was very much better, having had no breathlessness for over a fortnight and the cyanosis having quite passed off. The arteries felt much softer.

On October 2nd his vascular pressure was 150 mm. of Hg. He was discharged from hospital a week later, having had no cough or dyspnoea for over a month, and having gained 5 lbs. weight since his admission.

Six months later, he was re-admitted to hospital with a return of all his previous symptoms. His heart was considerably more enlarged than when he went out, but the sounds, though feeble, were both closed. His arteries were again thickened and tightened up, and the Sphygmomanometer showed a pressure of 195 mm. of Hg. Under the same treatment as before his vascular pressure again fell to 150 mm. of Hg. but no lower, and he was discharged his symptoms having completely disappeared.

In this case no amount of Erythrol would enable us entirely to do away with the thickening of the vessel wall or to bring the vascular pressure below 140 mm., and, on the second admission, below 150 mm. of Hg. This I think is explained by the fact that the patient's arteries showed a considerable amount of organic change, either fibrous or actually calcareous, but along with this there was present a hypertrophied muscular coat which could and did go into a state of hypertonus. This hypertonus we were able to get rid of, greatly to the patient's relief.

Case I, II, III, and IV. are further examples of this/

this state of affairs.

In the following case we have a similar condition present, but in this instance the arterial wall showed a tendency to go into a state of spasm, owing to which the vascular pressure ran up to 250 mm. of Hg. or, in some cases, as high as 290 mm. of Hg. Along with this the patient suffered from severe breathlessness and symptoms of cerebral angiospasm, the condition was immediately relieved by ¼ or ½ gr. of Erythrol Tetranitrate.

The case, which was very interesting, was as follows:-

Case XI. Female, aged 52, admitted to hospital with a history of cardiac deficiency extending over a period of six months. Six months before admission, she had a severe attack of cough, shortness of breath, and weakness of her legs. She recovered more or less from this, and three months later she had a similar attack. A month later she began to get decidedly worse, becoming very breathless on the slightest exertion. This breathlessness increased in intensity and began to trouble her even when she was in bed, until eventually she was practically never free from it. Along with this her legs began to swell, and she had frequent fits of dizziness. In addition to this she had occasional attacks of aphasia, during which she stammered and was unable to get her words out.

She gave a history of several attacks of acute Rheumatism, the first occurring when she was eleven years of age.

She stated that she had led a very hard life, often having/

having had very little food, and that she had taken a considerable quantity of alcohol.

On examination she looked very ill. She had a severe cough, was very short of breath and had a considerable amount pf oedema of her feet and ankles. Her face and lips were cyanotic and her finger-tips had a distinctly bluish tinge.

Her pulse was 84 per minute and was regular in time and force. The vessel wall was distinctly hard and thick, and her vascular pressure was 295 mm. of Hg.

Her heart was enlarged, the apex being situated in the sixth left intercostal space just internal to the nipple. Both sounds were closed in all areas and no murmurs were audible.

Her urine was acid, specific gravity 1017, contained 10 grs. of urea to the ounce but no abnormal constituents.

There was nothing particular to note in any of her other systems.

She was admitted on June 10th, and stated that, in addition to the signs and symptoms above noted, she at times lost control of power in her legs and feet and was also unable to speak. Her vascaulr pressure was 295 mm. of Hg., and in spite of her symptoms and the size of her heart, no cardiac valvular lesion could be made out.

On June 13th her vascular pressure was down to 215 mm. of Hg., and a well-marked presystolic murmur was heard in the mitral area. She was given Pot. Iodide.

On June 15th her pressure was down to 185 mm. of Hg., and a great improvement in her conditon was noticeable; she/

she said she felt better than she had done for a long time. Her arteries felt distinctly softer.

On June 25th her pressure was 220 mm. of Hg., and she was so far improved that she was allowed up.

On July 4th she was not feeling so well. She had a severe headache, was breathless, and had obvious difficulty with articulation. Her vascular pressure was 268 mm. of Hg. She was given Erythrol Tetranitrate gr. ½ with Manitol gr. ½. On the following day she was much better, and her pressure had fallen to 190 mm. of Hg.

The patient remained in hospital for five months and her condition showed a marked variability, some days she was fairly well, and on these occasions her pressure was always below 200 mm. of Hg. At other times, on the contrary, she complained of headaches, breathlessness, and various nervous phenomena, such as tinglings in her tongue, weakness in her limbs, and difficulty with her articulation. On these occasions her pressure was invariably above 250 mm. of Hg., and her symptoms were always relieved by ½ gr. of Erythrol Tetranitrate. During the whole of this time, however, her general condition showed a considerable improvement, her oedema and other signs of cardiac insufficiency disappeared, and with the exception of the attacks above referred to she was practically well.

The following extracts from the notes taken at the time will show clearly the kind of attack to which she was subject, and the marked effect of Erythrol Tetranitrate.

Aug. 30th./

Aug. 30th. Patient was particularly well this morning, but became restless about 6 p.m. By 7.30 p.m. she was complaining of violent pains in her head, was very breathless and had considerable difficulty with articulation. She spoke very slowly and seemed to make a great effort to get her words out, but her speech was very difficult to understand.
At 8.30 p.m. her vascular pressure was 260 mm. of Hg. and she was given ¼ gr. of Erythrol.
At 9.30 p.m. her pressure was 207 mm. of Hg., she was feeling drowsy, her discomfort had practically disappeared, and she went to sleep soon after.

Aug. 31st. Morning:- Her pressure was 175 mm. of Hg.
Evening:- Her pressure was 185 mm. of Hg.

Sept. 1st. Vascular pressure 204 mm. of Hg.

Sept.14th. Vascular pressure has been going down for some days and is now 182 mm. of Hg.

Sept.15th. 2.30 a.m. Patient had a bad attack of breathlessness with difficulty of speech and headache. Her radial and brachial arteries felt tightened up and her pressure was 250 mm. of Hg. She was given Erythrol gr. ¼.
3.0 a.m. Vascular pressure 174 mm. of Hg. Symptoms slightly relieved, vessels feel relaxed.
3.30 a.m. Vascular pressure 162 mm. of Hg. Breathing easier, short, deep, silent, inspiration followed by long, noisy expiration.
4.0 a.m. Vascular pressure 150 mm. of Hg. Patient quiet and breathing better.
11.0 p.m. Vascular pressure 182 mm. of Hg.

Sept.23rd. Vascular pressure has averaged 200 mm. of Hg. for last few days.
12 Midnight. Patient had slight attack, with pains in left side of body and left leg.
Vascular pressure 270 mm. of Hg., given Erythrol gr. ¼.
12.10 a.m. Vascular pressure 219 mm. of Hg., and patient perspiring profusely.
12.20 a.m. Pressure 202 mm. of Hg., patient feeling sleepy, and fell asleep a few minutes later.
11.0 a.m. Pressure 220 mm. of Hg. Patient feels quite well.

Oct. 26th. Patient complained of headache and was sick.
10.45 p.m. Breathlessness severe, vascular pressure 250 mm. of Hg., given Erythrol gr. ¼.
11.0 p.m. Pressure 196 mm. of Hg.
11.15 p.m. Pressure 150 mm. of Hg. Patient sleeping quietly.

During the last five weeks of her sojourn in hospital, she had no further attacks of angiospasm, and was discharged on December 2nd. apparently cured.

Eight months later she was readmitted with a return of all her previous symptoms. She was breathless, cyanosed, and oedmatous. Her pulse was 60 per minute, her respirations 20, her temperature 97·8°F., and her vascular pressure 200 mm. of Hg. Her arteries felt hard and resistant to pressure, and any advantage she had gained by her previous residence in hospital had quite disappeared. She again had violent attacks of dyspnoea as on the former occasion, of which I will quote two.

Aug. 31st. Severe attack of headache, nausea, and breathlessness.
At 1.45 a.m. her vascular pressure was 240 mm. of Hg. She was given Erythrol ½ gr.
1.50 a.m. Vascular pressure 230 mm. of Hg.
1.55 a.m. Vascular pressure 180 mm. of Hg., breathlessness greatly improved - feeling sleepy.
2.0 a.m. Vascular pressure 180 mm. of Hg. Patient sleeping quietly.

Sept.18th. 1.50 a.m. Patient breathing very badly, complaining of nausea and flushing of head. Vascular pressure 270 mm. of Hg., given Erythrol gr. ½.
1.55 a.m. Vascular pressure 228 mm. of Hg. Feeling easier.
2.0 a.m. Vascular pressure 210 mm. of Hg.
2.5 a.m. Vascular pressure 175 mm. of Hg. Feeling much relieved and going off to sleep.

After being in hospital for six wekks she was again discharged having had no angiospasmodic attacks during the last three weeks of her stay.

No comment on this case is necessary, it speaks for itself.

In this case more than any other, we see the remarkable/

remarkable effect of hypertonus and angiospasm on the Sphygmomanometer reading and the marked relief produced by a vaso-dilator.

In this case the action of Erythrol is clearly brought out; it produces its maximum effect half an hour after administration, and the effects have not quite worn off until about 24 hours later; therefore, if we give ¼ or ½ gr. of Erythrol morning and evening we can keep the tendency to hypertonus well under control.

The following case is similar to the preceding one, but not so marked.

Case XII. Male, aged 44. This was a case of Aortic Incompetence. He admitted having had syphilis at 27 years of age. On admission he was suffering from cough, dyspnoea, and oedema, and stated that he was subject to sudden attacks of very severe breathlessness and dizziness.

On examination his pulse was 80 per minute, his respirations 22, and his temperature 97·8°F.

His pulse was irregular, and his arterial walls felt abnormally thick and hard for a man of his age. His vascular pressure was 160 mm. of Hg. His heart was enlarged, the sounds were somewhat weak and a murmur of aortic incompetence was present.

His urine was acid, specific gravity 1020, and contained a trace of albumen but no blood or casts.

His other systems showed no abnormality.

He improved greatly under rest in bed and medicinal treatment. Almost every night for the first fortnight he had dyspnoeic attacks, during which he sat up in/

in bed and complained of a pain across his chest. These attacks were invariably relieved by Tabellae Trinitrini, and during the attack the vascular pressure was high. The following will show the nature of the attacks. -

Aug. 4th. 7.30 p.m. Very short of breath and sitting up in bed. Vascular pressure 178 mm. of Hg. and arteries felt abnormally tightened up. Given Tabellae Trinitrini. 8.0 p.m. Attack relieved, Pressure 140 mm. of Hg.

Aug. 5th. Vascular pressure 152 mm. of Hg.

Aug. 10th. Breathless attack, vascular pressure 172 mm. of Hg. Given Tabellae Trinitrini, attack relieved, and pressure fell to 138 mm. of Hg.

He had numerous similar attacks which were always relieved by the same remedy.

He was discharged, after having been in hospital for six weeks, greatly improved. During the last three weeks of his stay he was free from dyspnoea.

This concludes my series of cardiac cases which show a condition of Hypertonus, either in normal arteries or in those with hypertrophied coats, and which also show the marked beneficial result obtained by the administration of a vaso-dilator, the one chiefly used being Erythrol Tetranitrate.

This Hypertonus may be either continually present as in Cases I. II. & IV. to X., or it may be only spasmodic as in Cases III, XI & XII. In these cases where it is continuous, frequent small doses of Erythrol, or in some cases merely rest in bed and the removal as far as possible of any irritant in the blood, is sufficient to produce/

produce a marked and lasting effect. In the second type where the hypertonus is more of the nature of an angiospasm, a dose of the vaso-dilator as soon as the attack shows signs of coming on, and small preventative doses between the attacks, are sufficient.

In order that this condition of excessive contraction of arterial wall should occur we must either have a hypertrophied muscular coat present, or a coat of normal thickness which is in a specially irritable condition and in a state of excessive spasm. It may be objected that the contraction which is overcome by a vaso-dilator is not excessive but is merely the normal tonus of the vessel. In order to prove that this is not so, I made a series of observations on the vascular pressures of normal individuals after the administration of ½ gr. of Erythrol Tetranitrate. The conclusions I came to were as follows:-

In a youth of 20 years of age or thereabouts, in whom the arterial walls were quite normal, the fall in pressure which occurred during the first half-hour after the administration of gr. ½ of Erythrol was on an average 5 mm. of Hg., and that, moreover, the maximum fall occurred during the first five minutes, and after that the fall was inconsiderble.

In a man of 30 or thereabouts, the fall was larger in amount, being on an average from 10-14 mm. of Hg. during the first half hour after the drug had been given. Here again the maximum fall occurred during the first five minutes.

Above/

Above this age, the results, of course, varied according to the condition of the arteries.

From these results, I argued that if I obtained a fall of 30-50 mm. of Hg. or more after the administration of ½ gr. of Erythrol, I was dealing with an excessive degree of tonus in either a hypertrophied muscular coat or one of normal thickness; if by means of a vaso-dilator I was able to get the pressure down to subnormal or normal, I argued that no organic change had occurred and that the condition was entirely due to spasm, but, if, on the other hand, I was unable to reduce the pressure to normal, I came to the conclusion that there was some organic thickening of the vessel wall, in most cases probably muscular hypertrophy. One thing at any rate was certain, namely, that a muscular coat of considerable extent was present, and that it was in a condition to contract and relax to a marked degree, whatever other change might have occurred in the vessel wall. Even in arteries which showed a very marked degree of arteriosclerosis, a definite result was obtained by means of a vaso-dilator, thus proving clinically that Russell's observations on the pathology of arteriosclerosis were correct.

I will now give a few more cases which further illustrate the question of Hypertonus.

Case XIII. Male. Admitted to hospital with the history that 10 days previously he had a severe headache - worse on the left side - and along with it an increasing feeling of numbness and weakness over the right side of his/

his body. His arm was most affected; he had two or three giddy turns, and his speech was occasionally slightly affected.

He gave no history of syphilis. He had led a hard life, and was moderate in the use of alcohol and tobacco.

He had hemiparesis of his right side, but no abnormal sensory or vasomotor phenomena.

Pulse. Regular, synchronous, equal in force. Rise and fall slow, apex well sustained. Vessel wall thickened and tortuous, Vascular pressure 204 mm. of Hg.

Heart. Enlarged, impulse feeble, both sounds closed in all areas.

Urine. Acid, 1020, no abnormal constituents.

Progress and Treatment. (Extract from Case Book.)

June 19th. Vascular pressure 204 mm. of Hg.

" 20th. Given Pot. Iodide gr. v. T.I.D.

" 21st. Vascular pressure 180 mm. of Hg.

" 22nd. Pot. Iod. increased to gr. xv. T.I.D.

" 24th. Vascular pressure 142 mm. of Hg.

" 27th. Vascular pressure 158 mm. of Hg.
Pot. Iod. increased to gr. xx. T.I.D.
Erythrol Tetranitrate gr. ss. T.I.D.

" 29th. Vascular pressure 142 mm. of Hg.

July 3rd. Vascular pressure 115 mm. of Hg.
Erythrol stopped.

" 8th. Vascular pressure 132 mm. of Hg.

" 17th. Vascular pressure 170 mm. of Hg.
Erythrol gr. ½ T.I.D.

" 21st. Vascular pressure 122 mm. of Hg.

" 28th. Vascular pressure 138 mm. of Hg.

Comment. This case is interesting as showing the much more rapid effect of Erythrol, as compared with Pot. Iodide, on the arteries. It also shows how the pressure began to rise again after the Erythrol was stopped, and how it at once fell by 50 mm. of Hg. when the vasodilator was recommenced. The fall of 50 mm. which we see on July 21st is more than normal, so that I think we can explain the presence of 170 mm. of July 17th by the Hypertonic theory, especially as after the vasodilator the pressure fell to normal. I think the pressure of 204 mm. on admission was also hypertonic, and the same phenomenon, as applied to the cerebral vessels, accounts for the nervous symptoms. This is borne out by the fact that the hemiparesis cleared up under treatment. If the cerebral angiospasm were very severe, it would take some time for the circulation in the brain to be completely restored, and possibly a small amount of permanent damage might be done to the nervous tissue.

The following case is of a similar type.

Case XIV. Male, aged 49. Admitted to hospital complaining of giddiness of ten weeks' duration.

History. These attacks of giddiness came on quite suddenly, usually after stooping, about ten weeks before admission. He had three attacks in one day with loss of sensation in the left side of the body, the whole side, including face, trunk, and limbs, feeling quite cold and numb. He suffered from nausea but had no vomiting, and once fell. His sight was bad and he had slight headache./

headache. At the time of admission he was slightly improved, and was usually able to avert the attacks by lying down, when after 10 minutes he was quite well again. He had them on an average twice a day, and they began with a gripping sensation round the left upper arm. He drank and smoked a considerable amount.

Nervous System. He suffered from slight frontal headaches and also from pains shooting along the vertex. Apart from the symptoms mentioned in the history, he showed no abnormality of sensation or sensibility. He showed no loss of power, and his reflexes were normal.

His refraction was all but normal, but his vision varied curiously from 6/12 to 6/36., nothing definitely abnormal was found in the fundus.

No nystagmus or abnormality of pupil reflexes.

Had evidence of old standing middle ear disease, inner ear was not affected.

Circulatory System.

Pulse, regular, good volume, 96 per minute, pressure 140 mm. of Hg. Vessel walls irregularily thickened.

Heart. Pulsation visible in sixth interspace one inch outside nipple line.

Right border was $1\frac{3}{4}$ inches from mid-sternal line and apex was in sixth interspace $6\frac{1}{4}$ inches from midline and $1\frac{1}{2}$ inches outside nipple.

Both sounds closed in all areas.

Urine. Acid, 1025, amber, no abnormal constituents.

Treatment and Progress.

Jan. 24th. Given Pot. Iodide gr. x. T.I.D. V.P. 142 mm. of Hg.

Jan. 30th. Slight headache. V.P. 130 mm. of Hg.

Feb. 9th. V.P. 110 mm. of Hg. Great improvement.

Feb. 14th. Discharged:- regarded as a case of cerebral angiospasm which had been completely cured.

A curious factor in this case was the variability of the vision which oscillated between 6/12th and 6/36th. These two cases may I think be compared to migraine of which they illustrate a severe and persistent type. The Hypertonus occurred in the whole arterial system, hence the raised vascular pressure, but was most severe in certain of the cerebral vessels.

Migraine is more essentially an angiospasm of certain localised cerebral vessels without the general hypertonus; it is also much more transitory in the mild forms, the effects usually wearing off in the course of a few hours without any special treatment. The symptoms while they last may include abnormalities of sensibility and sensation, hemianopsia and aphasia, and are practically always followed by very severe hemicrania. The attacks, if severe, can undoubtedly be shortened by the administration of a vaso-dilator.

While I was serving as a Surgeon on board H.M.S. "Neptune" in October, 1914, I saw a very severe case of aphasia due to cerebral angiospasm. The man, who was a stoker, was seized with vomiting during the night, and in the morning his messmates discovered that he was quite unable to speak. When I saw him he looked very dazed, and was unable to tell his name, or, in fact, to speak/

speak at all. He was, however, able to understand what was said to him, and indicated that he had a severe pain on the left side of his head, extending from the frontal region to the occipital. He also conveyed the impression to one that he had a peculiar feeling in his right hand and arm. When he afterwards regained his speech he confirmed this impression, and said that his hand and arm felt weak and that a tingling sensation was present in them. There seemed to be actually some slight loss of power on that side. He could not write and was unable to protrude his tongue. He had no other loss of power or sensibility. He had no nystagmus or strabismus, and his pupils reacted normally to light and accommodation. His superficial and deep reflexes were somewhat exaggerated on both sides. He was put to bed, surrounded by hot bottles, given a purge and a hypodermic injection of nitro-glycerine. He soon fell asleep and when he awoke he was able to talk quite naturally, though still somewhat dazed. He said that he had never had a previous attack of this sort, and the only cause to which this attack could be attributed was the fact that the previous day he had eaten some tinned meat which had been sent to him, and which he had left open for some hours. I thought it was quite a possibility that some toxin might have been present in the meat, especially as the attack had been ushered in by vomiting. I at once suspected that this was a case of cerebral angiospasm, as I had seen a similar, though much more severe one, when I was resident House Physician in the Edinburgh Royal Infirmary. In this previous case, however, the patient

was unable to speak for 24 hours. He was able to understand what was said to him and to write; he had also had previous attacks of aphasia.

In the first of these cases, the general arterial system did not seem to the finger to be hypertonic. As we had not a Sphygmomanometer on board, I was unable to take a reading. In the second case the general systolic vascular pressure was not increased to any extent.

These four cases all seem to me to be examples of cerebral angiospasm, the first two in addition to some generalised hypertonus. They were all no doubt due to some toxin circulating in the blood, acting, perhaps, on arteries which were in an unduly irritable condition. What this toxin was it is impossible to say; it may have been introduced from without, but I think it was more probably an autointoxication from the intestinal tract. I am convinced that this is the condition of affairs which we meet with in migraine, but I think that the presence of the toxin is not the only factor necessary; in addition we require to have the vessels in a suitable condition to react to this unknown intoxication. Migraine very often seems to run in families, and the subject of this condition begins to suffer from it when quite young, and continues to do so till past middle life. Some people never suffer from it at all, while some are continually troubled with it. If we take it, as I think we may, that the same toxin is present in the body in both, some condition must be present in the arteries of the/

the one which renders them specially liable to be affected by this particular toxin. Much, however, can be done to counteract the tendency to vascular spasm by keeping the bowels in good order and the body generally in good condition, by getting plenty of fresh air and sleep, and avoiding, as far as possible, mental worry. If at any time the attacks of migraine become more frequent, ordinary tonic treatment will do much to diminish their frequency.

The following are the notes of some cases of nephritis with high blood pressures, and show the results of treatment with vaso-dilators.

Case XV. Male, aged 41. Subacute Nephritis.

This patient had suffered from headache and vomiting for four days prior to admission. His heart was slightly enlarged. His arteries felt hypertonic but did not show signs of arteriosclerosis or atheroma. His vascular pressure was 218 mm. of Hg.

Urine. Contained 1.8 grms. of albumen per oz. and granular and hyaline casts.

Four days before death he developed pneumonia, and oedema of the lungs and erysipelas of the face.

Progress and Treatment.

His vascular pressure was 218 mm. on admission; after a hot pack it fell to 210 mm. of Hg. Below this point it was impossible to bring the pressure in spite of most energetic treatment. He was given hot packs daily, drastic purging, Erythrol Tetranitrate, Liq. Trinitrini, and Pilocarpin, but all of no avail, the pressure/

pressure persistently remained at 210 mm. of Hg. or over. Finally he developed pneumonia.

Post-Mortem showed:-

(1) Pneumonia and oedema of the lungs.

(2) No hypertrophy, but dilatation of heart, papillary muscles fatty and cloudy.

(3) Marked subacute parenchymatous interstitial nephritis with rather coarse mottling. Capsule adherent and surface of kidney slightly granular.

(4) Spleen - acute congestion, enlarged and soft.

(5) Brain - very marked oedema.

Case XVI. Male, 59. Chronic Nephritis.

On admission, he complained greatly of headache, pains in abdomen, and difficulty in breathing. His legs and back were very oedematous. He gave a history of being in hospital on two or three occasions for the same condition.

Pulse. Regular, 100 per minute. Apex well sustained. Vascular pressure 202 mm. of Hg. Vessel wall much thickened.

Heart. Enlarged. Both sounds closed in all areas, second sound accentuated in aortic and pulmonary areas.

Lungs. Showed oedema of bases and some bronchitis.

Urine. Acid, 1020, amber, contained albumen.

Treatment and Progress. (Extract from Notes).

June. 29th. Pressure 202 mm. of Hg. Given purge (Jalap and Elaterium) and Erythrol.

" 30th. Pressure 176 mm. of Hg.

July 1st. Pressure 176 mm. of Hg. Headache much better

For the next month he continued to do quite well until July 28th when he developed a very bad headache and persistent vomiting, his vascular pressure was 255 mm. of Hg.

He was given a hot air bath which had only a very transitory effect; 16 ozs. of blood were removed from the arm, but this did not seem to have any beneficial action.

He was given Erythrol Tetranitrate gr.j. every two hours, at first he vomited it, but eventually he retained it. In the evening his vascular pressure was 240 mm. of Hg.

July 29th. He was rather better and did not vomit so much. He continued to have Erythrol gr. j.

July 30th. He was still better and was able to articulate better. Vascular pressure 214 mm. Erythrol reduced to gr.j. 4 hourly.

July 31st. Much improved, headache not so bad. Vascular pressure 210 mm.

For the next ten days he remained much the same. The pressure was usually about 220 mm. and he had no very severe headaches. At the end of this time the Erythrol was stopped. Two days later the pressure began to rise again and the heart began to show signs of serious failure. He was given digitaline but without effect. A 'pulsus alterans' set in and auricular fibrillation developed. The patient became very drowsy and finally sank into coma and died.

Post-Mortem showed:-

(1) Heart - hypertrophied, Coronary arteries showed patchy atheroma.

(2) Lungs - oedematous.

(3) Kidneys/

(3) Kidneys - Considerable narrowing of cortex and increase of peripelvic fat. Capsule adherent, and surface like morocco leather.

Microscopically, well-marked diffuse interstitial changes, vessels show an extreme degree of endarteritis deformans.

Case XVII. Female, aged 52. Chronic Interstitial Nephritis. The patient was admitted suffering from the effects of high vascular pressure.

She made little improvement and returned home at her own request.

On admission her pressure was 230 mm. of Hg. and she was treated by means of Nitrites and Pot. Iodide. Her pressure was brought down as low as 214 mm. of Hg., but beyond this point no treatment had any effect on it.

Case XVIII. Male, 46. Chronic Nephritis with Uraemia.

This patient was admitted complaining that for the three weeks before admission his eyesight had been gradually failing, being usually worst in the morning and tending to improve as the day went on. For the week previous to admission he had vomited once a day, the vomiting having no relation to food; and about this time he noticed that he had diplopia. He gave no venereal history, and was moderate in the use of alcohol and tobacco.

His pulse was regular, apex well sustained, Vascular pressure 195 mm. of Hg., and his vessel wall did not feel particularly thickened.

His/

His heart was much enlarged, the apex being situated in the sixth interspace 6½ inches from the mid-line. Both sounds were closed in all areas.

His eyes showed neuroretinitis with numerous old and recent retinal haemorrhages.

His kidneys were not palpable or tender.

His urine was acid, 1024, amber, and contained 3·9 grs. of albumen to the oz.

Progress and Treatment. On admission his vascular pressure was 195 mm. of Hg.; it soon rose to 200 mm. and 210 mm. and persistently remained there, all attempts to bring it down by vaso-dilators (Liquor Trinitrini), Hot Air baths, &c., having failed. He passed very little urine, and in spite of energetic treatment, developed uraemia on the third day, and died on the sixth day.

Case XIX. Female, aged 52. Chronic Interstitial ~~Pyelo-~~ Nephritis ~~litis~~ and Acute ~~Nephritis~~ Pyelitis.

She was admitted to hospital with the history that for the last year she had been troubled with severe occipital headaches, and that her eyesight had been gradually failing. Ten weeks before admission she was seized with a severe fit of vomiting and pain in the epigastric region extending round to the lumbar regions. This had continued more or less up till the time of admission, the vomiting having no relation to food. She stated that she had known for more than two years that her kidneys were not right.

Her/

Her pulse was regular, apex well sustained. The vascular pressure was 190 mm. of Hg. and the vessel wall was thickened.

The heart was hypertrophied and a mitral systolic murmur was present.

Her abdomen was rigid and tender.

She had considerable dyspnoea and was breathing very deeply, but no abnormal physical signs were present in her chest.

Kidneys. Both her lumbar regions were markedly tender on pressure.

Her urine was acid, 1009, lemon, and contained albumen, red blood corpuscles, and casts.

Progress and Treatment. Her pressure persistently remained high, i.e., 190 mm. to 200 mm., in spite of treatment. She showed no improvement, and a week after admission pulmonary congestion set in and she died.

Post-Mortem showed:-

Heart. All cavities hypertrophied, especially left ventricle which was thickened and pale. Chronic thickening of mitral valve was present.

Aorta. Atheromatous, especially descending and abdominal aorta.

Lungs. Congestion and oedema.

Kidneys. Considerably reduced in size. Characteristic appearance of chronic interstitial nephritis. Both pelves show haemorrhage and blood clot in interior. Substance of kidney soft, capsule strips easily but leaves rough surface behind.

Microscopically, diffuse overgrowth of fibrous tissue/

tissue, vessels show marked thickening of intima leading to an extreme degree of narrowing. Marked catarrhal condition of tubules. Many of glomeruli reduced to fibrous knots.

Brain. Atheromatous patches in vessels at base. Albuminuric retinitis with scattered haemorrhages.

Case XX. Female, 55. Chronic Nephritis.

The patient was admitted to hospital with a long history of congestive attacks of the head, during which she felt as if her head and face were going to burst. Along with this her eyesight was failing, and for the last month before admission, she had noticed that oedema of her eyelids and ankles was present in the mornings and that she became breathless on exertion. For some years she had been troubled with frontal headaches.

Her pulse was regular, slow rise and fall, apex well sustained; her vascular pressure was 202 mm. of Hg., and the vessel walls felt somewhat thickened and hypertonic.

Her heart was slightly enlarged and both sounds were closed in all areas.

Her kidneys were neither tender or palpable, and her urine was acid and contained a large amount of urates and some albumen.

Progress and Treatment. She remained in hospital for three weeks. Her pressure at times rose to 210 mm. of Hg., and in spite of treatment by Erythrol Tetranitrate and Pot. Iodide it never fell below 190 mm. of Hg., her symptoms, however, improved.

These six cases all had vascular pressures of 200 mm. of Hg. or over. They were all treated by Erythrol Tetranitrate, and not one of them responded in the same way that the cardiac cases did. In spite of large doses of Erythrol, hot air baths, &c., not only did the pressure not fall, but in some cases actually continued to rise. In those which did respond the fall in pressure was practically negligible. One was tempted to suppose that in these nephritic cases the raised pressure was not hypertonic in character. In the following case, however, it was hypertonic, and although Erythrol had only a slight effect on it, yet one drug had a very marked effect.

Case XXI. Female, 48. Chronic Nephritis.

She was admitted to hospital complaining of severe headaches chiefly frontal in type, dyspnoea when tired, oedema of eyelids in mornings, and failing eyesight.

Pulse was regular in rate and rhythm, slow rise and fall, small amplitude. Vascular pressure 262 mm. of Hg. Vessel walls felt thickened and hypertonic.

Heart was somewhat enlarged, outwards, i.e., apex 5¾ inches from mid-line. Both sounds closed in all areas.

Kidneys were neither tender or palpable.

Urine. Neutral, 1012, pale yellow, contained albumen.

Treatment and Progress.

She was given Pot. Iodide, occasional doses of Erythrol Tetranitrate, and hot air baths. Under this treatment/

treatment during her first fortnight in hospital, her pressure fell from 262 mm to 230 mm., below this point, however, it would not fall.

Her pressure then suddenly rose to 264 mm., and it was decided to try the effects of Veratrine. The result was as follows:-

10.15 p.m.	Vascular pressure 264 mm. of Hg.
10.20 p.m.	Given 1 cc. of Veratrine hypodermically.
10.35 p.m.	Vascular pressure 134 mm. of Hg.
10.50 p.m.	" " 146 " " "
11.5 p.m.	" " 148 " " "
11.20 p.m.	" " 164 " " "
11.35 p.m.	" " 178 " " "
11.50 p.m.	" " 170 " " "
12.20 a.m.	" " 148 " " "
1.55 a.m.	" " 152 " " "
11.5 a.m.	" " 218 " " "
10.0 p.m.	" " 256 " " "

A few minutes after the injection she developed severe vomitng and sweating, which persisted for two hours. She was still sick next morning, and was not herself for practically three days. The injection was given immediately after a hot air bath, which failed to lower the pressure or produce sweating to any great extent. Two days after her pressure was 282 mm. and the following day 252 mm.

Finally she was discharged slightly improved. She still had headaches and her pressure was very variable.

In this case Erythrol and hot air baths had practically no effect on the pressure, but the effect of Veratrine was somewhat startling. The pressure fell 130 mm. of Hg. in the course of a few minutes. The effect on the patient, however, was somewhat severe. It is quite likely that with smaller doses we may here have a valuable/

valuable therapeutic agent for lowering the excessively high pressure got in nephritis.

This shows that we have in nephritis either a toxin different from that which causes the hypertonus in chronic heart cases, or else it is present in such excessive quantities that nothing short of a poisonous dose of Erythrol will affect it. It is a well-known pharmacological fact that the various drugs which cause the contraction or relaxation of muscle, produce their effects by acting on different sections of the neuro-muscular mechanism. Some act on the nerve cells, some on the axis cylinders, some on the neuro-muscular junction, and some on the actual muscle fibres themselves. In like manner it is not only possible but probable that, of the toxins which cause the contraction of the walls of bloodvessels, some act on the vaso-motor nerve cells in the brain, some act on the nerve fibres, some act on the neuro-muscular junction in the vessel wall, and some act on the actual muscle fibre of the wall itself. In order, therefore, to counteract the action of any toxin, we must have a drug which acts on the same section of the neuro-muscular mechanism, or on a section on the distal side of that acted on by the toxin. To neutralise a toxin, therefore, which has for its effect the stimulation of the neuro-muscular junction, we must have a drug which counteracts this stimulation either at the neuro-muscular junction or at the muscle fibre itself; one which acts on either the nerve fibres or the nerve cells will have no effect. The nitrites produce their effect by paralysing the neuro-

-muscular junction, if, therefore we have a vaso-constriction produced by the action of a toxin on the muscular element of the vessel wall, the administration of the nitrite group will have no effect on it; we must give a drug which paralyses the muscle fibre itself.

Up to the present the actual toxin which causes the hypertonus in nephritis has not been isolated. Some authorities think that it is epinephrin, while others think that some waste product is the cause. If we presume that the toxin, whatever it is, produces hypertonus by its action on the muscle elements of the wall, we have an explanation of the failure of the nitrites to produce any beneficial result; I only refer, however, to the hypertonus which occurs in well advanced cases of subacute and chronic nephritis, and not to that which by many authorities is said to be a precusor of chronic nephritis and which responds well to the nitrite group.

Conclusions.

I. The vessel wall when thickened and hardened by arteriosclerosis opposes a very definite resistance to compression, and, therefore, gives rise to an increase in the Sphygmomanometer reading obtained.

II. Actual degeneration is not the only factor which increases the resistance of the vessel wall, but hypertrophy of the tunica media and hypertonus or spasm of a normal or hypertrophied muscular coat, also produces a definite resistance to compression.

The term "Hypertonus" may be used for the condition where/

where the vessel walls are continually in a condition of increased tonus, while angiospasm may be limited to the acute exacerbation of spasm; both conditions thicken the wall and diminish the lumen.

III. Hypertonus is present in many cases of chronic myocarditis. The effect is to raise the peripheral resistance, and embarrass the heart's action, and so bring about heart failure.

IV. By administering a vaso-dilator to these cases, along with a cardiac tonic, very marked beneficial effects can be obtained. In these cases the actual blood-pressure in the peripheral arteries is not high, the Sphygmomanometer gives us in addition to blood pressure a measure of the resistance of the vessel wall.

V. The actual error due to Hypertonus varies from 30 mm. to 100 mm. of Hg. The average error in my cases was about 55 mm. of Hg.

In nephritis cases the average amount I was able to lower the pressure by means of Erythrol was 26 mm. of Hg. as compared with 55 mm. in cardiac cases, though the original pressure in the nephritis cases was much higher.

The following is a table of my results:-

TABLE/

Sex of Patient.	Age of Patient.	Maximum Pressure.	Minimum Pressure.	Amount which Pressure fell.
Male.	57	200	180	20
"	68	210	166	44
"	45	200	154	46
"	40	182	112	70
"	36	160	120	40
"	38	162	122	40
"	65	170	135	35
"	67	218	146	72
"	64	200	140	60
Female.	52	295	150	145
Male.	44	178	140	38
"	49	140	110	32
"	66	172	146	26
"	70	238	198	40
"	?	204	115	90
	NEPHRITIS CASES.			
Male.	59	255	210	45
*Female.	48	264	134	130*
"	55	208	190	18
"	52	230	214	16
"	52	200	178	22
Male.	72	170	140	30

*Pressure lowered by 1 cc. of Veratrine hypodermically.

VI. Hypertonus and angiospasm are probably due to some toxin circulating in the blood, probably in many cases derived from the gastro-intestinal tract. The Hypertonus of Nephritis is due to a different toxin from that causing/

causing angiospasm and the hypertonus met with in cardiac cases. The toxin met with in nephritis probably acts directly on the muscle fibres of the vessel wall, while that met with in cardiac cases, &c., produces its effects by acting on the neuro-muscular junction.

Erythrol Tetranitrate which also acts on the neuro-muscular junction has a beneficial effect in the second type of case, but not in those of nephritic origin.

VII. The Hypertonus, not due to nephritis, is very amenable to treatment if treated early enough; if allowed to progress, however, it leads to hypertrophy and arterio-sclerosis.

VIII. Erythrol Tetranitrate is a very good vaso-dilator to use in cases of hypertonus. It produces its effect in less than 15 minutes, and the vaso-dilatation produced lasts for 12 hours or more. The most suitable dose to give is gr. ss. to gr. j.; if the desired result is not obtained by gr. j., repeated if necessary, no effect is likely to be got by increasing the dose. It should be given night and morning, as by this method the hypertonus is kept continually under control; if the spasm is very severe it may be given three times a day.

In cardiac cases with hypertonus Erythrol in gr. ss. doses acts very well as a hypnotic; it is quite safe and seems to have no depressant action of the heart.

Erythrol does not overcome the hypertonus of nephritis; this, however, may be counteracted by Veratrine, but its action is somewhat violent.

IX./

IX. Various nervous symptoms may be caused by angio-spasm, i.e., migraine, and may or may not be associated with general hypertonus.

The cases quoted were with one or two exceptions under my care while a resident House Physician in the Royal Infirmary, and I convey my grateful thanks to Professor Russell for permitting me to utilise them for the purposes of this Thesis.

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